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<input type="checkbox"/>	L1	(NGF-activated protein kinase)	0

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☐ 1. Document ID: US 20020160933 A1

Using default format because multiple data bases are involved.

L3: Entry 1 of 3

File: PGPB

Oct 31, 2002

PGPUB-DOCUMENT-NUMBER: 20020160933

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20020160933 A1

TITLE: Methods and compositions for producing a neurosalutary effect in a subject

PUBLICATION-DATE: October 31, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Benowitz, Larry I.	Newton Centre	MA	US	

US-CL-CURRENT: 514/1

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	RMC	Draw Desc
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☐ 2. Document ID: US 20020160933 A1

L3: Entry 2 of 3

File: DWPI

Oct 31, 2002

DERWENT-ACC-NO: 2003-328371

DERWENT-WEEK: 200331

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TITLE: Producing neurosalutary effect, and treating neurological disorder, in a subject, by administering a therapeutically effective amount of a compound that modulates the activity of N-kinase, to the subject

INVENTOR: BENOWITZ, L I

PRIORITY-DATA: 2001US-0949200 (September 7, 2001), 2000US-0656915 (September 7, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
<u>US 20020160933 A1</u>	October 31, 2002		020	A61K031/00

INT-CL (IPC): A61 K 31/00

ABSTRACTED-PUB-NO: US20020160933A

BASIC-ABSTRACT:

NOVELTY - Producing (M1) a neurosalutary effect in a subject, and treating a subject

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suffering from neurological disorder, involves administering a therapeutically effective amount of a compound (I) that modulates the activity of N-kinase, to the subject.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

- (1) identifying (M2) a compound capable of producing a neurosalutary effect in a subject, by contacting N-kinase or its biologically active fragment, with a test compound and determining the ability of the test compound to modulate the activity of N-kinase;
- (2) a compound capable of producing a neurosalutary effect in a subject identified by the above method;
- (3) an isolated N-kinase polypeptide (II) of the type that:
  - (a) is present in neonatal brain tissue
  - (b) is inhibited in the presence of 6-thioguanine
  - (c) is activated in the presence of Mn+2 but not by Mg+2 or Ca+2
  - (d) has a molecular weight of 49 kDa, and
  - (e) is eluted from a Cibacron Blue column at a NaCl concentration of 1.5-1.75 M;
- (4) an antibody which is specifically reactive with an epitope of (II);
- (5) a fragment of (II) comprising at least 15 contiguous amino acids, and capable of eliciting an immune response; and
- (6) an isolated nucleic acid molecule (III) encoding a polypeptide comprising a sequence of 272 amino acids fully defined in the specification.

ACTIVITY - Anticonvulsant; Cerebroprotective; Neuroprotective; Nootropic.

No supporting biological data is given.

MECHANISM OF ACTION - Modulator of N-kinase activity (claimed); Promotes neuronal survival, axonal outgrowth and neuronal regeneration; Intracellular mediator of axonal outgrowth.

No supporting biological data is given.

USE - M1 is useful for producing a neurosalutary effect, and thus for treating a subject e.g. mammal, preferably human, suffering from neurological disorder such as spinal cord injury (including monoplegia, diplegia, paraplegia, hemiplegia and quadriplegia), epilepsy, stroke and Alzheimer's disease. The treatment method further involves making a first assessment of a nervous system function prior to administering (I) and making a second assessment of a nervous system function after administering (I) to the subject. The nervous system function is a sensory function, cholinergic innervation or vestibulomotor function (claimed).

(II) is useful as bait protein in a two- or three-hybrid assay, to identify other proteins, which bind to or interact with N-kinase.

Full	Title	Citation	Front	Review	Classification	Date	Reference			Claims	KBNC	Draw Desc
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☐ 3. Document ID: JP 2004523470 W, WO 200220056 A2, AU 200187118 A, EP 1315514 A2

DERWENT-ACC-NO: 2002-393816

DERWENT-WEEK: 200451

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TITLE: Producing a neurosalutary effect in a subject e.g., one suffering from neurological disorder such as stroke, to treat the subject, by administering a compound that modulates activity of N-kinase

INVENTOR: BENOWITZ, L I

PRIORITY-DATA: 2000US-0656915 (September 7, 2000)

## PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
<u>JP 2004523470 W</u>	August 5, 2004		077	A61K045/00
<u>WO 200220056 A2</u>	March 14, 2002	E	042	A61K045/00
<u>AU 200187118 A</u>	March 22, 2002		000	A61K045/00
<u>EP 1315514 A2</u>	June 4, 2003	E	000	A61K038/18

INT-CL (IPC): A61 K 9/10; A61 K 9/127; A61 K 38/18; A61 K 45/00; A61 P 9/10; A61 P 9/12; A61 P 25/00; A61 P 25/02; A61 P 25/08; A61 P 25/14; A61 P 25/16; A61 P 25/18; A61 P 25/24; A61 P 25/28; A61 P 43/00; C07 K 14/475; C07 K 16/40; C12 N 9/12; C12 N 15/09; C12 Q 1/48; G01 N 33/15; G01 N 33/50; G01 N 33/53; G01 N 33/566

ABSTRACTED-PUB-NO: WO 200220056A

## BASIC-ABSTRACT:

NOVELTY - Producing (M1) a neurosalutary effect in a subject e.g., a subject suffering from a neurological disorder, to treat the subject suffering from the neurological disorder, involving administering to the subject a compound (I) that modulates the activity of N-kinase, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

- (1) an isolated N-kinase polypeptide (II) of the type that: is present in neonatal brain tissue; is inhibited in the presence of 6-thioguanine; is activated in the presence of Mn<sup>2+</sup>, but not by Mg<sup>2+</sup> or Ca<sup>2+</sup>; has a molecular weight of approximately 49 kDa; and is eluted from a Cibacron Blue column at a sodium chloride concentration of 1.5-1.75 M;
- (2) an antibody (III) which is specifically reactive with an epitope of (II);
- (3) a fragment (IV) of (I), which comprises at least 15 contiguous amino acids, and is able to elicit an immune response;
- (4) an isolated nucleic acid molecule that encodes (II); and
- (5) a compound capable of producing a neurosalutary effect in a subject identified using (II).

ACTIVITY - Nootropic; neuroprotective; cerebroprotective; anticonvulsant; vulnerary; tranquilizer; antiparkinsonian; antimanic; antidepressant.

MECHANISM OF ACTION - N-kinase activity modulator; neuronal survival modulator; neuronal regeneration modulator; neuronal axonal outgrowth of central nervous system neurons e.g., retinal ganglion cells, modulator (all claimed).

No data given.

USE - (I) is useful for producing a neurosalutary effect in a subject e.g., a subject suffering from a neurological disorder, to treat the subject (preferably, humans)

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suffering from the neurological disorder. The neurosalutary effect is produced by modulating neuronal survival, modulating neuronal regeneration or modulating neuronal axonal outgrowth of central nervous system neurons e.g., retinal ganglion cells, in a subject suffering from a neurological disorder such as spinal cord injury characterized by monoplegia, diplegia, paraplegia, hemoplegia and quadriplegia, or suffering from epilepsy, stroke or Alzheimer's disease.

(II) is useful for identifying a compound capable of producing a neurosalutary effect in a subject, preferably a compound which inhibits or stimulates the activity of N-kinase, which involves contacting (II) or its biologically active fragment with a test compound and determining the ability of the test compound to modulate the activity of N-kinase, thereby identifying a compound capable of producing a neurosalutary effect in a subject. The ability of the test compound to modulate the activity of N-kinase is determined by assessing the ability of the test compound to modulate N-kinase-dependant phosphorylation of a substrate. Optionally, (I) is identified using (II) by the following method which involves contacting (II) or its biologically active fragment, with a test compound, an N-kinase substrate (e.g., histone HF-1 protein), radioactive ATP (preferably gamma -32P), and Mn2+; and determining the ability of the test compound to modulate N-kinase dependent phosphorylation of the substrate, thereby identifying a compound capable of producing a neurosalutary effect in a subject. (II) used in the methods described above is preferably a recombinantly produced human N-kinase. Optionally, (II) is bovine N-kinase purified from a bovine source. The methods further involve determining the ability of the test compound to modulate axonal outgrowth of central nervous system neuron (all claimed).

(M1) is useful for treating a neurological disorder such as dementia's related to Alzheimer's disease, Parkinson's disease, senile dementia, Huntington's disease, Creutzfeldt-Jakob disease, Korsakoff's psychosis, mania, anxiety disorders, obsessive-compulsive disorder, anxiety, bipolar affective disorder. The methods are useful for preventing or treating neurological deficits in embryos or fetuses in utero, in premature infants, or in children with need of such treatment, including those with neurological birth defects. (I) is also useful for modulating activity of N-kinase, in vitro to modulate axonal outgrowth in vitro.

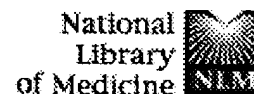
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N-kinase	3

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☐ **1:** Mochizuki N, Yamashita S, Kurokawa K, Ohba Y, Nagai T, Miyawaki A, Matsuda M. Related Articles, Li

**Spatio-temporal images of growth-factor-induced activation of Ras and Rap1.**  
Nature. 2001 Jun 28;411(6841):1065-8.  
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☐ **2:** Minneman KP, Lee D, Zhong H, Berts A, Abbott KL, Murphy TJ. Related Articles, Li

**Transcriptional responses to growth factor and G protein-coupled receptors in PC12 cells: comparison of alpha(1)-adrenergic receptor subtypes.**  
J Neurochem. 2000 Jun;74(6):2392-400.  
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**ErbB-4 activation promotes neurite outgrowth in PC12 cells.**  
J Neurochem. 2000 Mar;74(3):979-87.  
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☐ **4:** Mahata SK, Mahata M, Wu H, Parmer RJ, O'Connor DT. Related Articles, Li

**Neurotrophin activation of catecholamine storage vesicle protein gene expression: signaling to chromogranin a biosynthesis.**  
Neuroscience. 1999 Jan;88(2):405-24.  
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☐ **5:** Swanson KD, Taylor LK, Haung L, Burlingame AL, Landreth GE. Related Articles, Li

**Transcription factor phosphorylation by pp90(rsk2). Identification of Fos kinase and NGFI-B kinase I as pp90(rsk2).**  
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J Biol Chem. 1998 Sep 18;273(38):24624-32.  
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☐ **7:** Grimes ML, Beattie E, Mobley WC. Related Articles, Li

**A signaling organelle containing the nerve growth factor-activated receptor tyrosine kinase, TrkA.**  
Proc Natl Acad Sci U S A. 1997 Sep 2;94(18):9909-14.  
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☐ **8:** Xia Z, Dickens M, Raingeaud J, Davis RJ, Greenberg ME. Related Articles, Li

**Opposing effects of ERK and JNK-p38 MAP kinases on apoptosis.**  
Science. 1995 Nov 24;270(5240):1326-31.  
PMID: 7481820 [PubMed - indexed for MEDLINE]

Volonte C, Greene LA.

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 Nerve growth factor-activated protein kinase N modulates the cAMP-dependent protein kinase.

J Neurosci Res. 1995 Jan 1;40(1):108-16.

PMID: 7714918 [PubMed - indexed for MEDLINE]


 10: [Loeb DM, Tsao H, Cobb MH, Greene LA.](#)

[Related Articles, Li](#)


 NGF and other growth factors induce an association between ERK1 and the NGF receptor, gp140prototr.

Neuron. 1992 Dec;9(6):1053-65.

PMID: 1463607 [PubMed - indexed for MEDLINE]


 11: [Volonte C, Greene LA.](#)

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 Nerve growth factor-activated protein kinase N. Characterization and rapid near homogeneity purification by nucleotide affinity-exchange chromatography.

J Biol Chem. 1992 Oct 25;267(30):21663-70.

PMID: 1400478 [PubMed - indexed for MEDLINE]


 12: [Volonte C, Greene LA.](#)

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 6-Methylmercaptopurine riboside is a potent and selective inhibitor of nerve growth factor-activated protein kinase N.

J Neurochem. 1992 Feb;58(2):700-8.

PMID: 1309569 [PubMed - indexed for MEDLINE]


 13: [Volonte C, Rukenstein A, Loeb DM, Greene LA.](#)

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 Differential inhibition of nerve growth factor responses by purine analogues: correlation with inhibition of a nerve growth factor-activated protein kinase.

J Cell Biol. 1989 Nov;109(5):2395-403.

PMID: 2553745 [PubMed - indexed for MEDLINE]

 14: [Rowland EA, Muller TH, Goldstein M, Greene LA.](#)

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 Cell-free detection and characterization of a novel nerve growth factor-activated protein kinase in PC12 cells.

J Biol Chem. 1987 Jun 5;262(16):7504-13.

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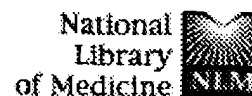
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## Cell-free detection and characterization of a novel nerve growth factor-activated protein kinase in PC12 cells.

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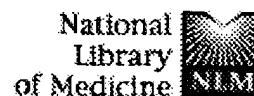
We have developed a cell-free assay to detect and characterize nerve growth factor (NGF)-activated protein kinase activity. Cultured PC12 cells were briefly exposed to NGF, and extracts of these were assayed for phosphorylating activity using exogenously added tyrosine hydroxylase as substrate. Tyrosine hydroxylase was employed since it is an endogenous substrate of NGF-regulated kinase activity and is activated by phosphorylation. In the cell-free assay, extracts prepared from NGF-treated cells yielded a 2-3-fold greater incorporation of phosphate into tyrosine hydroxylase as compared with extracts of control, NGF untreated cells. Activation did not occur, however, if NGF was added directly to cell extracts. The NGF-stimulated phosphorylating activity appeared to be due to regulation of a protein kinase rather than of a phosphoprotein phosphatase. Characterization of the kinase (designated as kinase N) showed that it is soluble, is detectably activated within 1-3 min after cells are exposed to NGF and maximally activated by 10 min, is half-maximally activated with 0.5 nM NGF and maximally activated with 1 nM NGF, is detectable in the presence of either  $Mg^{2+}$  or  $Mn^{2+}$  but does not require  $Ca^{2+}$ , does not require nonmacromolecular cofactors, can use histone H1 as a substrate, and exhibits a 2-fold increase in apparent  $V_{max}$  in response to NGF but does not undergo a significant change in apparent  $K_m$  for either ATP or GTP. A number of characteristics of kinase N were assessed including susceptibility to inhibitors, substrate specificity, cofactor requirements, ATP dependence, and lack of down-regulation by prolonged exposure to a phorbol ester. These studies indicated that it lacks tyrosine kinase activity and is distinct from a variety of well-characterized protein kinases including cAMP-dependent protein kinase, protein kinase C ( $Ca^{2+}$ /phospholipid dependent enzyme),  $Ca^{2+}$ /calmodulin-dependent kinase, and casein kinase II. Preliminary purification data show that the kinase has a basic pI and that it has an apparent  $M_r$  of 22,000-25,000. The only amino acid in tyrosine hydroxylase found to be phosphorylated by the semipurified kinase is serine.

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## Nerve growth factor-activated protein kinase N. Characterization and rapid near homogeneity purification by nucleotide affinity-exchange chromatography.

Volonte C, Greene LA.

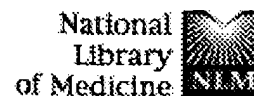
Department of Pathology, College of Physicians and Surgeons, Columbia University, New York, New York 10032.

Protein kinase N (PKN) is a protein kinase rapidly activated by nerve growth factor (NGF) and other agents in PC12 pheochromocytoma and additional cell types. PKN is selectively inhibited by purine analogs, and this property has served both as a diagnostic for PKN activity and to establish its apparent involvement in certain pathways of the NGF mechanism of action. The present work has focused on further characterization, identification, and purification of NGF-activated PKN. We show here that PKN can be substantially enriched by elution from ion exchange resins with ATP. We exploited this novel technique (nucleotide affinity exchange chromatography) to devise two alternative isolation schemes for PKN. One utilizes sequential chromatographic steps and provides preparation that is apparently 60% homogeneous for PKN and represents a total enrichment of approximately 10,000-fold. The other is a single column procedure and includes prewashes with NAD. This method yields material that is about 5-10% homogeneous for PKN, requires about 1 h, and can be applied to multiple samples in parallel. The ATP elution technique furthermore distinguishes NGF-regulated from basal PKN activity and thereby suggests the presence of distinct PKN isoforms. The applications of sucrose gradient centrifugation, gel filtration chromatography, sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE)/silver staining, affinity labeling with 8-azido-ATP/SDS-PAGE, autophosphorylation (after SDS-PAGE, blotting and renaturation) all indicate PKN has an apparent molecular mass of 45-47 kDa and is mainly monomeric in solution. These and additional properties appear to distinguish PKN from many previously described protein kinases.

PMID: 1400478 [PubMed - indexed for MEDLINE]

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## Nerve growth factor-activated protein kinase N modulates the cAMP-dependent protein kinase.

Volonte C, Greene LA.

Department of Pathology, College of Physicians and Surgeons of Columbia University, New York, New York.

Protein kinase N (PKN) is a serine/threonine protein kinase rapidly activated by nerve growth factor (NGF) and other agents in various cell lines. The possible involvement of PKN in the multiple pathways of the NGF mechanism of action was previously established through the use of purine analogs, some of which are apparently specific inhibitors of this kinase. Since a PKN-like activity is modulated in several cell lines by cAMP analogs and this activation requires the activity of cAMP-dependent protein kinase, the aim of the present work is to investigate possible interactions between PKN and C-PKA. Pre-incubation of the two kinases in the presence of ATP leads to potentiated phosphorylation of histone H1, Kemptide (a substrate for C-PKA, but not for PKN), and several additional substrates. This augmented phosphorylating activity is insensitive to thioguanine (an inhibitor for PKN, but not for C-PKA) and is suppressed both by the Walsh inhibitor and by the regulatory subunit of PKA. PKN-pretreated C-PKA shows a significant decrease in  $K_m$  for Kemptide and a substantial increase in  $V_{max}$ . C-PKA and PKN are widely expressed enzymes and the possibility of PKN-dependent modulation of PKA in intact cells would therefore have biological implications for signal transduction mechanisms.

PMID: 7714918 [PubMed - indexed for MEDLINE]

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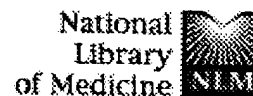
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## A signaling organelle containing the nerve growth factor-activate receptor tyrosine kinase, TrkA.

Grimes ML, Beattie E, Mobley WC.

Department of Biochemistry, Massey University, Palmerston North, New Zealand. M.L.Grimes@Massey.ac.NZ

The topology of signal transduction is particularly important for neurons. Neurotrophic factors such as nerve growth factor (NGF) interact with receptor distal axons and a signal is transduced by retrograde transport to the cell body ensure survival of the neuron. We have discovered an organelle that may acco for the retrograde transport of the neurotrophin signal. This organelle is derive from endocytosis of the receptor tyrosine kinase for NGF, TrkA. In vitro reactions containing semi-intact PC12 cells and ATP were used to enhance recovery of a novel organelle: small vesicles containing internalized NGF bow to activated TrkA. These vesicles were distinct from clathrin coated vesicles, uncoated primary endocytic vesicles, and synaptic vesicles, and resembled transport vesicles in their sedimentation velocity. They contained 10% of the t bound NGF and almost one-third of the total tyrosine phosphorylated TrkA. These small vesicles are compelling candidates for the organelles through whi the neurotrophin signal is conveyed down the axon.

PMID: 9275225 [PubMed - indexed for MEDLINE]

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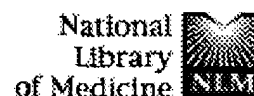
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
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
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


















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
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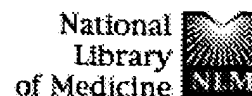
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## Nerve growth factor (NGF) responses by non-neuronal cells: detection by assay of a novel NGF-activated protein kinase.

Volonte C, Greene LA.

Department of Pathology, College of Physicians and Surgeons, Columbia University, New York, New York 10032.

Past work described the partial purification and characterization of a novel serine protein kinase activity designated protein kinase N (PKN) that is activated by nerve growth factor (NGF) in cultured PC12 cells [Rowland et al. (1987) J. Biol. Chem. 262; 7504-7513]. We have now devised a rapid, sensitive technique for partially purifying and assaying PKN activity in cell extracts. This methodology was applied to the IARC-EW-1 osteosarcoma and several additional non-neuronal cell lines that possess NGF receptors but that lack both morphological and a variety of additional biochemical responses to NGF. In each case, NGF significantly elevated PKN activity. The assay also revealed activation of PKN activity in IARC-EW-1 cells by additional agents, including epidermal growth factor, fibroblast growth factor, phorbol ester, and a cAMP analog. Also tested were an NGF-receptor-deficient PC12 cell variant and sublines thereof into which human NGF receptors had been introduced [Hempstead et al. (1989) Science 247:373-375]. Acquisition of the NGF receptors resulted in NGF-activatable PKN activity. These findings indicate that detection of PKN activity may serve as a sensitive means to test NGF responsiveness in cells lacking macroscopic responses to the factor and that non-neuronal cells may be useful for studying primary signaling events in the NGF mechanism of action.

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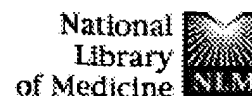
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
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
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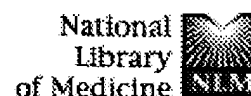
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## jun-NH2-terminal kinase activation mediated by UV-induced DNA lesions in melanoma and fibroblast cells.

Adler V, Fuchs SY, Kim J, Kraft A, King MP, Pelling J, Ronai Z.

Molecular Carcinogenesis Program, American Health Foundation, Valhalla, N York 10595, USA.

jun-NH2-terminal kinase (JNK) belongs to a family of protein kinases that phosphorylates c-Jun, ATF2, and Elk1 in response to various forms of stress including UV irradiation and heat shock. Although in previous studies we have demonstrated the importance of membrane components for JNK activation by irradiation, here we have elucidated the role of DNA damage in this response. show that in vitro-irradiated or sonicated DNA that is added to proteins prepared from UV-treated cells can further induce JNK activation in a dose-dependent manner. When compared with UV-B (300 nm), UV-C (254 nm), which is better absorbed by the DNA, is significantly more potent in activating JNK. Furthermore, when wavelengths lower than 300 nm were filtered out, UV-B was no longer able to activate JNK. With the aid of melanoma and fibroblast cells, which exhibit different resistances to irradiation and require different UV doses to generate the same number of DNA lesions, we demonstrate that above a threshold level of 0.45 lesions and up to 0.75 lesions per 1875 bp, the degree of JNK activation correlates with the amount of lesions induced by UV-C irradiation. Finally, to explore the role of nuclear and mitochondrial DNA (mtDNA) in mediating JNK activation after UV irradiation, we have used cells that lack mtDNA. Although the lack of mtDNA did not impair the ability of UV to activate JNK, when enucleated, these cells had lost the ability to activate JNK in response to UV irradiation. Overall, our results suggest that DNA damage in the nuclear compartment is an essential component that acts in concert with membrane-anchored proteins to mediate c-Jun phosphorylation by JNK.

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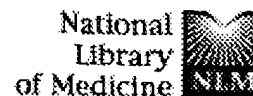
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## UV irradiation and heat shock mediate JNK activation via altern: pathways.

Adler V, Schaffer A, Kim J, Dolan L, Ronai Z.

Molecular Carcinogenesis Program, American Health Foundation, Valhalla, N York 10595, USA.

To elucidate cellular pathways involved in Jun-NH2-terminal kinase (JNK) activation by different forms of stress, we have compared the effects of UV irradiation, heat shock, and H<sub>2</sub>O<sub>2</sub>. Using mouse fibroblast cells (3T3-4A) we show that while H<sub>2</sub>O<sub>2</sub> is ineffective, UV and heat shock (HS) are potent induc of JNK. The cellular pathways that mediate JNK activation after HS or UV exposure are distinctly different as can be concluded from the following observations: (i) H<sub>2</sub>O<sub>2</sub> is a potent inhibitor of HS-induced but not of UV-indu JNK activation; (ii) Triton X-100-treated cells abolish the ability of UV, but not HS, to activate JNK; (iii) the free radical scavenger N-acetylcysteine inhibits U but not HS-mediated JNK activation; (iv) N-acetylcysteine inhibition is blocke by H<sub>2</sub>O<sub>2</sub> in a dose-dependent manner; (v) a Cockayne syndrome-derived cell exhibits JNK activation upon UV exposure, but not upon HS treatment. The significance of Jun phosphorylation by JNK after treatment with UV, HS, or H<sub>2</sub>O<sub>2</sub> was evaluated by measuring Jun phosphorylation in vivo and also its binding activity in gel shifts. HS and UV, which are potent inducers of JNK, increased the level of c-Jun phosphorylation when this was measured by [32P] orthophosphate labeling of 3T3-4A cultures. H<sub>2</sub>O<sub>2</sub> had no such effect. Althou, H<sub>2</sub>O<sub>2</sub> failed to activate JNK in vitro and to phosphorylate c-Jun in vivo, all th forms of stress were found to be potent inducers of binding to the AP1 target sequence. Overall, our data indicate that both membrane-associated compon and oxidative damage are involved in JNK activation by UV irradiation, where HS-mediated JNK activation, which appears to be mitochondrial-related, utiliz cellular sensors.

PMID: 7592807 [PubMed - indexed for MEDLINE]

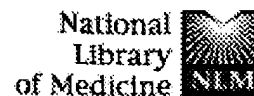
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**A peptide encoding the c-Jun delta domain inhibits the activity of a c-jun amin**  
**terminal protein kinase.**

J Biol Chem. 1994 Apr 15;269(15):11186-91.

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☐ 2: Franklin CC, Unlap T, Adler V, Kraft AS.

Related Articles, Li

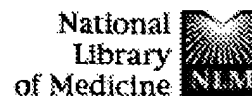
**Multiple signal transduction pathways mediate c-Jun protein phosphorylation.**

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**A peptide encoding the c-Jun delta domain inhibits the activity of c-jun amino-terminal protein kinase.****Adler V, Unlap T, Kraft AS.**

Division of Hematology/Oncology, University of Alabama, Birmingham 3529

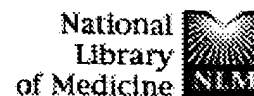
Evidence suggests that the c-Jun protooncogene delta (delta) domain (amino acids 31-60) helps regulate the transcriptional activating capacity of c-Jun by modulating the amino-terminal phosphorylation of this protein. By using a peptide encoding the delta domain and purified amino-terminal c-Jun protein kinase, we demonstrate that the delta domain peptide inhibits phosphorylation of the amino terminus of both c-Jun and the related protein JunD. The delta domain peptide inhibited the activation of the c-Jun amino-terminal protein kinase by phorbol esters in permeabilized U937 leukemic cells. Mutation of c-Jun followed by transfection into U937 leukemic cells demonstrated that partial deletions of the delta domain are sufficient to block phosphorylation of the amino terminus of c-Jun. In vitro deletion of the amino-terminal (amino acids 31-44) half of the delta domain inhibited the phosphorylation of c-Jun. However, deletion of the carboxyl-terminal (amino acids 45-60) half only partially inhibited c-Jun phosphorylation. Therefore, these results indicate that the delta domain sequence is an important regulator of c-Jun amino-terminal phosphorylation.

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☐ 4: [Adler M, Wagner G.](#)

[Related Articles, Li](#)

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Biochemistry. 1992 Feb 4;31(4):1031-9.  
PMID: 1734953 [PubMed - indexed for MEDLINE]

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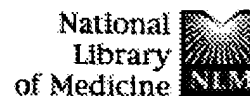
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## Affinity-purified c-Jun amino-terminal protein kinase requires serine/threonine phosphorylation for activity.

Adler V, Polotskaya A, Wagner F, Kraft AS.

Division of Hematology/Oncology, University of Alabama, Birmingham 3529

The addition of phorbol esters to U937 leukemic cells stimulates the phosphorylation of c-Jun on serines 63 and 73. To isolate the protein kinase which stimulates this phosphorylation, we have used heparin-Sepharose chromatography followed by affinity chromatography over glutathione-Sepharose beads bound with a fusion protein of glutathione S-transferase and amino acid: 89 of c-Jun (GST-c-Jun). Using this procedure we purify a 67-kDa protein which is capable of phosphorylating GST-c-Jun as well as the complete c-Jun protein. By making mutations in serines 63 and 73 and then creating a fusion protein with GST (GST-c-Jun mut), we demonstrate that this protein kinase specifically phosphorylates these sites in the c-Jun amino terminus. Treatment of purified c-Jun amino-terminal protein kinase (cJAT-PK) with phosphatase 2A inhibits its ability to phosphorylate GST-c-Jun. This inactivated enzyme can be reactivated by phosphorylation with protein kinase C (PKC), although PKC is not capable of phosphorylating the GST-c-Jun substrate. Because v-Jun cannot be phosphorylated in vivo, we compared the ability of cJAT-PK to bind to GST-v-Jun or GST-c-Jun mut. The cJAT-PK bound 50-fold better to GST-c-Jun mut than GST-v-Jun suggesting that the delta domain which is missing in v-Jun plays a role in binding the cJAT-PK. These results suggest that there is a protein kinase cascade mediated by protein phosphatases and PKC which regulates c-Jun phosphorylation.

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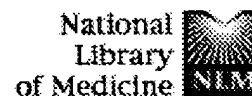
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

















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



















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













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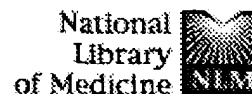
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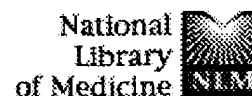
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## Identification of a human brain-specific isoform of mammalian STE20-like kinase 3 that is regulated by cAMP-dependent protein kinase.

Zhou TH, Ling K, Guo J, Zhou H, Wu YL, Jing Q, Ma L, Pei G.

Shanghai Institute of Cell Biology, Chinese Academy of Sciences, 320 Yue Y Road, Shanghai 200031, People's Republic of China.

A novel isoform of mammalian STE20-like kinase 3 (MST3) with a different coding region from MST3, termed MST3b, was identified by searching through expressed sequence tag data base and obtained by rapid amplification of cDNA 5'-ends. MST3b was assigned to the long arm of human chromosome 13, D13S159-D13S280, by use of the National Center for Biotechnology Information sequence-tagged sites data base. Reverse transcription-polymerase chain reaction and Northern blot analysis with a probe derived from 5' distinct sequence of MST3b revealed that the expression of MST3b mRNA is restricted to the brain in contrast to ubiquitous distribution of MST3 transcript. Western analysis confirmed the brain-specific expression of MST3b protein. In situ hybridization of rat brain sections with a MST3b-specific probe indicated that MST3b is widely expressed in different brain regions, with especially high expression in hippocampus and cerebral cortex. When expressed in human embryonic kidney 293 (HEK293) cells, MST3b effectively phosphorylated myelin basic protein, well as undergoing autophosphorylation. Interestingly, expression of MST3, but not MST3b, in HEK293 cells was able to activate the endogenous p42/44 mitogen-activated protein kinase (MAPK) up to 4-fold, whereas neither isoform activated p38 MAPK under the same conditions. Further experiments demonstrated that MST3b, but not MST3, was effectively phosphorylated by activation of cyclic AMP-dependent protein kinase (PKA) in both in vivo and vitro assays. The mutation of Thr-18 into Ala in MST3b (T18A), a putative PK phosphorylation site that is absent in MST3, abolished its phosphorylation by PKA. Consequently, expression of the T18A mutant in HEK293 cells led to partial activation of p42/44 MAPK, indicating that MST3b is under the regulation of PKA. Taken together, our data provide evidence that the two isoforms of STE20-like kinase 3 are differentially distributed and regulated.

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DN PREV199598224193  
TI \*\*\*Nerve\*\*\* \*\*\*growth\*\*\* \*\*\*factor\*\*\* - \*\*\*activated\*\*\*  
\*\*\*protein\*\*\* \*\*\*kinase\*\*\* N modulates the cAMP-dependent protein  
kinase.  
AU Volonte, C. [Reprint author]; Greene, L. A.  
CS Inst. Neurobiol., CNR, Viale Marx 15, 00137 Rome, Italy  
SO Journal of Neuroscience Research, (1995) Vol. 40, No. 1, pp. 108-116.  
CODEN: JNREDK. ISSN: 0360-4012.  
DT Article  
LA English  
ED Entered STN: 23 May 1995  
Last Updated on STN: 9 Jun 1995

L4 ANSWER 4 OF 23 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on  
STN DUPLICATE 3  
AN 1995:224826 BIOSIS  
DN PREV199598239126  
TI Stimulation of vgf gene expression by NGF is mediated through multiple  
signal transduction pathways involving protein phosphorylation.  
AU Salton, Stephen R. J. [Reprint author]; Volonte, Cinzia; D'Arcangelo,  
Gabriella  
CS Fishberg Res. Cent. Neurobiol., Mt. Sinai Sch. Med., Box 1065, One Gustave  
Levy Place, New York, NY 10029-6574, USA  
SO FEBS Letters, (1995) Vol. 360, No. 2, pp. 106-110.  
CODEN: FEBLAL. ISSN: 0014-5793.  
DT Article  
LA English  
ED Entered STN: 31 May 1995  
Last Updated on STN: 11 Jul 1995

L4 ANSWER 5 OF 23 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on  
STN DUPLICATE 4  
AN 1993:412748 BIOSIS  
DN PREV199396078473  
TI A purine analog-sensitive protein kinase activity associates with Trk  
nerve growth factor receptors.  
AU Volonte, Cinzia [Reprint author]; Loeb, David M.; Greene, Lloyd A.  
CS Inst. Neurobiol., CNR, Viale Marx, 15, 00156 Rome, Italy  
SO Journal of Neurochemistry, (1993) Vol. 61, No. 2, pp. 664-672.  
CODEN: JONRA9. ISSN: 0022-3042.  
DT Article  
LA English  
ED Entered STN: 8 Sep 1993  
Last Updated on STN: 3 Jan 1995

L4 ANSWER 6 OF 23 CAPLUS COPYRIGHT 2004 ACS on STN  
AN 1993:248429 CAPLUS  
DN 118:248429  
TI Association of a purine-analog-sensitive protein kinase activity with p75  
nerve growth factor receptors  
AU Volonte, Cinzia; Ross, Alonzo H.; Greene, Lloyd A.  
CS Coll. Physicians Surg., Columbia Univ., New York, NY, 10032, USA  
SO Molecular Biology of the Cell (1993), 4(1), 71-8  
CODEN: MBCEEV; ISSN: 1059-1524  
DT Journal

LA English

L4 ANSWER 7 OF 23 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on  
STN DUPLICATE 5

AN 1993:7506 BIOSIS

DN PREV199395007506

TI \*\*\*Nerve\*\*\* \*\*\*growth\*\*\* \*\*\*factor\*\*\* - \*\*\*activated\*\*\*  
\*\*\*protein\*\*\* \*\*\*kinase\*\*\* N: Characterization and rapid near  
homogeneity purification by nucleotide affinity exchange chromatography.

AU Volonte, Cinzia [Reprint author]; Greene, Lloyd A.

CS Institute Neurobiology, CNR, Viale Marx 15, 00156 Rome, Italy

SO Journal of Biological Chemistry, (1992) Vol. 267, No. 30, pp. 21663-21670.  
CODEN: JBCHA3. ISSN: 0021-9258.

DT Article

LA English

ED Entered STN: 10 Dec 1992  
Last Updated on STN: 13 Dec 1992

L4 ANSWER 8 OF 23 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on  
STN DUPLICATE 6

AN 1992:145774 BIOSIS

DN PREV199293079999; BA93:79999

TI 6 METHYLMERCAPTOPYRINE RIBOSIDE IS A POTENT AND SELECTIVE INHIBITOR OF  
\*\*\*NERVE\*\*\* \*\*\*GROWTH\*\*\* \*\*\*FACTOR\*\*\* - \*\*\*ACTIVATED\*\*\*  
\*\*\*PROTEIN\*\*\* \*\*\*KINASE\*\*\* N.

AU VOLONTE C [Reprint author]; GREENE L A

CS DEP PATHOLOGY, COLLEGE PHYSICIANS SURGEONS COLUMBIA UNIVERSITY, 630 WEST  
168TH STREET, NEW YORK, NY 10032, USA

SO Journal of Neurochemistry, (1992) Vol. 58, No. 2, pp. 700-708.  
CODEN: JONRA9. ISSN: 0022-3042.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 12 Mar 1992  
Last Updated on STN: 13 Mar 1992

L4 ANSWER 9 OF 23 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.  
on STN DUPLICATE 7

AN 91102585 EMBASE

DN 1991102585

TI Cick sympathetic neurons in culture respond differentially to nerve growth  
factor and conditioned medium from activated splenic lymphocytes.

AU Luo J.-J.; Hasegawa S.

CS Center for Neurobiology, and Molecular Immunology, Chiba University Sch.  
of Med., Inohana 1-8-1, Chiba 280, Japan

SO Neuroscience Research, (1991) 10/2 (137-148).  
ISSN: 0168-0102 CODEN: NERADN

CY Ireland

DT Journal; Article

FS 021 Developmental Biology and Teratology  
037 Drug Literature Index

LA English

SL English

L4 ANSWER 10 OF 23 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on  
STN DUPLICATE 8

AN 1990:379641 BIOSIS

DN PREV199090066322; BA90:66322

TI INDUCTION OF ORNITHINE DECARBOXYLASE BY NERVE GROWTH FACTOR IN PC12 CELLS  
DISSECTION BY PURINE ANALOGUES.

AU VOLONTE C [Reprint author]; GREENE L A

CS DEP PATHOL, CENT NEUROBIOL BEHAVIOUR, COLL PHYSICIANS SURGEONS, COLUMBIA  
UNIV, NEW YORK, NY 10032, USA

SO Journal of Biological Chemistry, (1990) Vol. 265, No. 19, pp. 11050-11055.  
CODEN: JBCHA3. ISSN: 0021-9258.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 21 Aug 1990  
Last Updated on STN: 22 Aug 1990

L4 ANSWER 11 OF 23 CAPLUS COPYRIGHT 2004 ACS on STN

AN 1990:152533 CAPLUS

DN 112:152533

TI Nerve growth factor stimulates a protein kinase in PC-12 cells that  
phosphorylates microtubule-associated protein-2

AU Miyasaka, Tadayo; Chao, Moses V.; Sherline, Peter; Saltiel, Alan R.  
CS Lab. Mol. Oncol., Rockefeller Univ., New York, NY, 10021, USA  
SO Journal of Biological Chemistry (1990), 265(8), 4730-5  
CODEN: JBCHA3; ISSN: 0021-9258  
DT Journal  
LA English

L4 ANSWER 12 OF 23 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on  
STN  
AN 1991:149500 BIOSIS  
DN PREV199140069105; BR40:69105  
TI MOLECULAR CHARACTERISTICS OF AN \*\*\*NGF\*\*\* - \*\*\*ACTIVATED\*\*\*  
\*\*\*PROTEIN\*\*\* \*\*\*KINASE\*\*\* PKN.  
AU VOLONTE C [Reprint author]; GREENE L A  
CS DEP PATHOL, COLUMBIA UNIV, NEW YORK, NY 10032, USA  
SO Society for Neuroscience Abstracts, (1990) Vol. 16, No. 1, pp. 825.  
Meeting Info.: 20TH ANNUAL MEETING OF THE SOCIETY FOR NEUROSCIENCE, ST.  
LOUIS, MISSOURI, USA, OCTOBER 28-NOVEMBER 2, 1990. SOC NEUROSCI ABSTR.  
ISSN: 0190-5295.  
DT Conference; (Meeting)  
FS BR  
LA ENGLISH  
ED Entered STN: 23 Mar 1991  
Last Updated on STN: 22 May 1991

L4 ANSWER 13 OF 23 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on  
STN DUPLICATE 9  
AN 1990:130545 BIOSIS  
DN PREV199089069356; BA89:69356  
TI MULTIPLE PATHWAYS OF N KINASE ACTIVATION IN PC12 CELLS.  
AU ROWLAND-GAGNE E [Reprint author]; GREENE L A  
CS DEPARTMENT PATHOLOGY, COLUMBIA UNIVERSITY, 630 WEST 168 STREET, NEW YORK,  
NY 10032, USA  
SO Journal of Neurochemistry, (1990) Vol. 54, No. 2, pp. 424-433.  
CODEN: JONRA9. ISSN: 0022-3042.  
DT Article  
FS BA  
LA ENGLISH  
ED Entered STN: 13 Mar 1990  
Last Updated on STN: 13 Mar 1990

L4 ANSWER 14 OF 23 CANCERLIT on STN DUPLICATE 10  
AN 90132665 CANCERLIT  
DN 90132665 PubMed ID: 2153751  
TI Multiple pathways of N-kinase activation in PC12 cells.  
AU Rowland-Gagne E; Greene L A  
CS Department of Pharmacology, New York University School of Medicine.  
NC GM 07238 (NIGMS)  
NS16036 (NINDS)  
SO JOURNAL OF NEUROCHEMISTRY, (1990 Feb) 54 (2) 423-33.  
Journal code: 2985190R. ISSN: 0022-3042.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS MEDLINE; Priority Journals  
OS MEDLINE 90132665  
EM 199002  
ED Entered STN: 19941107  
Last Updated on STN: 19970509

L4 ANSWER 15 OF 23 CANCERLIT on STN DUPLICATE 11  
AN 90248158 CANCERLIT  
DN 90248158 PubMed ID: 2159763  
TI Nerve growth factor (NGF) responses by non-neuronal cells: detection by  
assay of a novel \*\*\*NGF\*\*\* - \*\*\*activated\*\*\* \*\*\*protein\*\*\*  
\*\*\*kinase\*\*\*.  
AU Volonte C; Greene L A  
CS Department of Pathology, College of Physicians and Surgeons, Columbia  
University, New York, New York 10032.  
NC NS16036 (NINDS)  
SO GROWTH FACTORS, (1990) 2 (4) 321-31.  
Journal code: 9000468. ISSN: 0897-7194.  
CY Switzerland  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS MEDLINE; Priority Journals



OS MEDLINE 90248158  
EM 199006  
ED Entered STN: 19941107  
Last Updated on STN: 19970509

L4 ANSWER 16 OF 23 CANCERLIT on STN  
AN 90657021 CANCERLIT  
DN 90657021  
TI THE CHARACTERIZATION, PARTIAL PURIFICATION AND REGULATION OF AN  
\*\*\*NGF\*\*\* - \*\*\*ACTIVATED\*\*\* \*\*\*PROTEIN\*\*\* \*\*\*KINASE\*\*\* IN PC12  
CELLS.

AU Gagne E R  
CS New York Univ., NY.  
SO Diss Abstr Int [B], (1989) 49 (9) 3551.  
ISSN: 0419-4217.  
DT (THESIS)  
LA English  
FS Institute for Cell and Developmental Biology  
EM 198912  
ED Entered STN: 19941107  
Last Updated on STN: 19970509

L4 ANSWER 17 OF 23 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on  
STN DUPLICATE 12  
AN 1990:30235 BIOSIS  
DN PREV199089017201; BA89:17201  
TI DIFFERENTIAL INHIBITION OF NERVE GROWTH FACTOR RESPONSES BY PURINE  
ANALOGUES CORRELATION WITH INHIBITION OF A \*\*\*NERVE\*\*\* \*\*\*GROWTH\*\*\*  
\*\*\*FACTOR\*\*\* - \*\*\*ACTIVATED\*\*\* \*\*\*PROTEIN\*\*\* \*\*\*KINASE\*\*\* .

AU VOLONTE C [Reprint author]; RUKENSTEIN A; LOEB D M; GREENE L A  
CS DEP PATHOL, COLL PHYSICIANS SURG COLUMBIA UNIV, NEW YORK 10032, USA  
SO Journal of Cell Biology, (1989) Vol. 109, No. 5, pp. 2395-2404.  
CODEN: JCLBA3. ISSN: 0021-9525.  
DT Article  
FS BA  
LA ENGLISH  
ED Entered STN: 19 Dec 1989  
Last Updated on STN: 20 Dec 1989

L4 ANSWER 18 OF 23 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation.  
on STN  
AN 89:568995 SCISEARCH  
GA The Genuine Article (R) Number: AX799  
TI DIFFERENTIAL INHIBITION OF NERVE GROWTH-FACTOR RESPONSES BY PURINE ANALOGS  
- CORRELATION WITH INHIBITION OF A \*\*\*NERVE\*\*\* \*\*\*GROWTH\*\*\* -  
\*\*\*FACTOR\*\*\* \*\*\*ACTIVATED\*\*\* \*\*\*PROTEIN\*\*\* - \*\*\*KINASE\*\*\*

AU VOLONTE C (Reprint); RUKENSTEIN A; LOEB D M; GREENE L A  
CS COLUMBIA UNIV COLL PHYS & SURG, DEPT PATHOL, NEW YORK, NY, 10032  
(Reprint); COLUMBIA UNIV COLL PHYS & SURG, CTR NEUROBIOL & BEHAV, NEW  
YORK, NY, 10032  
CYA USA  
SO JOURNAL OF CELL BIOLOGY, (1989) Vol. 109, No. 5, pp. 2395-2403.  
DT Article; Journal  
FS LIFE  
LA ENGLISH  
REC Reference Count: 41

L4 ANSWER 19 OF 23 CAPLUS COPYRIGHT 2004 ACS on STN  
AN 1989:434215 CAPLUS  
DN 111:34215  
TI The characterization, partial purification, and regulation of an  
\*\*\*NGF\*\*\* - \*\*\*activated\*\*\* \*\*\*protein\*\*\* \*\*\*kinase\*\*\* in PC12  
cells

AU Gagne, Elizabeth Rowland  
CS New York Univ., New York, NY, USA  
SO (1988) 166 pp. Avail.: Univ. Microfilms Int., Order No. DA8825019  
From: Diss. Abstr. Int. B 1989, 49(9), 3551-2  
DT Dissertation  
LA English

L4 ANSWER 20 OF 23 DISSABS COPYRIGHT (C) 2004 Proquest Information and  
Learning Company; All Rights Reserved on STN  
AN 88:18581 DISSABS Order Number: AAR8825019  
TI THE CHARACTERIZATION, PARTIAL PURIFICATION AND REGULATION OF AN  
\*\*\*NGF\*\*\* - \*\*\*ACTIVATED\*\*\* \*\*\*PROTEIN\*\*\* \*\*\*KINASE\*\*\* IN PC12  
CELLS

AU GAGNE, ELIZABETH ROWLAND [PH.D.]; GREENE, LLOYD A. [advisor]  
 CS NEW YORK UNIVERSITY (0146)  
 SO Dissertation Abstracts International, (1988) Vol. 49, No. 9B, p. 3551.  
 Order No.: AAR8825019. 166 pages.  
 DT Dissertation  
 FS DAI  
 LA English  
 ED Entered STN: 19921118  
 Last Updated on STN: 19921118

L4 ANSWER 21 OF 23 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on  
 STN DUPLICATE 13  
 AN 1987:340022 BIOSIS  
 DN PREV198784048965; BA84:48965  
 TI CELL-FREE DETECTION AND CHARACTERIZATION OF A NOVEL \*\*\*NERVE\*\*\*  
 \*\*\*GROWTH\*\*\* \*\*\*FACTOR\*\*\* - \*\*\*ACTIVATED\*\*\* \*\*\*PROTEIN\*\*\*  
 \*\*\*KINASE\*\*\* IN PC12 CELLS.  
 AU ROWLAND E A [Reprint author]; MUELLER T H; GOLDSTEIN M; GREENE L A  
 CS DEP PHARMACOL, NEW YORK UNIV SCH MED, NEW YORK, NY 10016, USA  
 SO Journal of Biological Chemistry, (1987) Vol. 262, No. 16, pp. 7504-7513.  
 CODEN: JBCHA3. ISSN: 0021-9258.  
 DT Article  
 FS BA  
 LA ENGLISH  
 ED Entered STN: 8 Aug 1987  
 Last Updated on STN: 8 Aug 1987

L4 ANSWER 22 OF 23 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on  
 STN  
 AN 1985:191693 BIOSIS  
 DN PREV198529081689; BR29:81689  
 TI CELL-FREE CHARACTERIZATION OF A \*\*\*NERVE\*\*\* \*\*\*GROWTH\*\*\*  
 \*\*\*FACTOR\*\*\* - \*\*\*ACTIVATED\*\*\* \*\*\*PROTEIN\*\*\* \*\*\*KINASE\*\*\* .  
 AU MULLER T H [Reprint author]; ROWLAND E A; GOLDSTEIN M; GREENE L A  
 CS DEP PHARMACOLOGY, NEW YORK UNIV SCH MED, 550 FIRST AVE, NEW YORK, NY  
 10016, USA  
 SO Biological Chemistry Hoppe-Seyler, (1985) Vol. 366, No. 4, pp. 323.  
 Meeting Info.: SYMPOSIUM ON SELECTED TOPICS OF NEUROBIOCHEMISTRY HELD AT  
 THE 36TH MOSBACHER MEETING OF THE GESELLSCHAFT FUER BIOLOGISCHE CHEMIE  
 (SOCIETY FOR BIOCHEMISTRY), APR. 18-20, 1985. BIOL CHEM HOPPE-SEYLER.  
 CODEN: BCHSEI. ISSN: 0177-3593.  
 DT Conference; (Meeting)  
 FS BR  
 LA ENGLISH

L4 ANSWER 23 OF 23 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation.  
 on STN  
 AN 85:231664 SCISEARCH  
 GA The Genuine Article (R) Number: AFR23  
 TI CELL-FREE CHARACTERIZATION OF A NERVE GROWTH-FACTOR ( \*\*\*NGF\*\*\* )-  
 \*\*\*ACTIVATED\*\*\* \*\*\*PROTEIN\*\*\* - \*\*\*KINASE\*\*\*  
 AU MULLER T H (Reprint); ROWLAND E A; GOLDSTEIN M; GREENE L A  
 CS NYU, DEPT PHARMACOL, NEW YORK, NY, 10016  
 CYA USA  
 SO BIOLOGICAL CHEMISTRY HOPPE-SEYLER, (1985) Vol. 366, No. 4, pp. 323.  
 DT Conference; Journal  
 FS LIFE  
 LA ENGLISH  
 REC Reference Count: 5  
 STN INTERNATIONAL LOGOFF AT 14:24:56 ON 05 NOV 2004